time of incubation. The curve appears to be characterized by two rates of fixation, one of about 0.021 μg of nitrogen per liter per day during the first 58 hours, the other of about 0.068 μg of nitrogen per liter per day during the rest of the experiment (*6*).

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5. A detailed description of our entire procedure is in preparation.
6. We wish to express our appreciation to R. H. Burris of the department of biochemistry, University of Wisconsin, and Richard Abrams of the research laboratory, Montefiore Hospital, Pittsburgh, for the advice they have generously given and for the use of the mass spectrometers in their laboratories. Both spectrometers are Consolidated-Nier isotope ratio machines. This research was supported by the University of Kentucky Faculty Research Fund and by the National Institutes of Health. The Alaska Department of Fish and Game provided laboratory facilities at the Kitoi Bay Research Station on Afognak Island.

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Sensory Deprivation and Visual Speed: An Analysis

Abstract. Speeds of moving objects were markedly underestimated by human observers after prolonged patternless visual stimulation. Even greater underestimation followed exposure to a "noisy" visual field; on the other hand, exposure to a hyperstable field caused overestimation. The effects of external visual noise simulate those of deprivation; this finding suggests that similarly disordered but spontaneous neural discharge dominates the visual nervous system in deprivation.

Experiments in sensory deprivation have been made to explore the consequences of prolonged exposure to visual fields in which normal spatiotemporal variation is sharply reduced. Results of experiments performed at McGill University (*1*) suggest that the apparent speed of a moving line markedly changes as one consequence of long-term exposure to an illuminated but patternless visual field. After such deprivation, observers reported that a straight black line slowly rotating against a dimly lit screen looked S-shaped. This distortion was attributed to an induced "perceptual lag": the ends of the line lag behind the center.

In preliminary investigations we found no evidence for a perceived distortion of the line. Instead, the apparent speed of the entire line was clearly reduced. Consequently, in further investigations we have used an apparatus (Fig. 1) designed to measure and thus quantify changes in apparent speed (*2*). The observer was instructed to fixate a black dot 1 m distant at eye level. He then saw a black bar (Fig. 1, *A*) sweep, like the second hand of a clock, through 90 deg from a horizontal to a vertical position, at a rate of 60 deg/sec. At the vertical, the bar disappeared from the observer's sight behind a screen. His task was to judge when the now-hidden bar would reach a fixed marker (Fig. 1, *B*) 10 deg beyond. This judgment was based on the observer's estimate of the speed of the bar during its visible 90 deg of travel. When he judged that the hidden bar had traversed the 10 deg to reach the marker, he stopped the coupled time clock (Fig. 1) which gave a measure of the apparent speed of the bar. The average of the initial judgments made by practiced observers was very close to the actual travel time. Twenty trials per observer were made before and after each experimental exposure.

In a first test to evaluate our interpretation of the McGill results, seven observers were deprived of patterned vision for 8 hours under conditions similar to those of the McGill study (*3*). The observers, lying on cots, were subjected to a masking acoustic noise at the maximum tolerable level; they wore tubes over their arms and hands and translucent goggles over their eyes. The goggles provided only a homogeneously illuminated, nonpatterned field of vision. After deprivation, each of the seven observers judged the speed of the sweep to be less (by an average of 16 percent) than he had initially judged it to be. The observers showed no accompanying change in simple motor reaction time. Having demonstrated and measured this consequence of generalized deprivation, we proceeded to further analyses.

Each of 12 observers was instructed to fix his gaze on the center of a patternless but illuminated circular field (intensity 0.2 ml) subtending 43 visual degrees in an otherwise dark field (patternless exposure field). Although only visual input was controlled, a significant reduction in apparent speed was produced with only ½ hour of exposure (Table 1). This test demonstrates that control of extravisual stimulation is not essential for obtaining the change in apparent speed.

Table 1. Changes in apparent speed after ½ hour of exposure. Exposure fields: H, hyperstable; D, dark; P, patternless; N, noisy.

Exposure field	Change in speed (%)	Significance* of differences between conditions		
H	+14.8	H–D: .05	H–P: .01	H–N: .001
D	− 4.8	D–P: N.S.†	D–N: .02	
P	−10.1	P–N: N.S.†		
N	−19.6			

* Significance levels derived from an analysis of variance. † Not significant.

Comparable experimental situations were then used to explore the effects of exposing the same observers to three other test fields devised on the basis of the following speculation. In the absence of normal stimulation coming from patterned fields, there is reason to suspect that the visual nervous system exhibits spontaneous and patternless activity (*4*). Dominance of the visual system for long periods by such intrinsic noise may result in reduction of

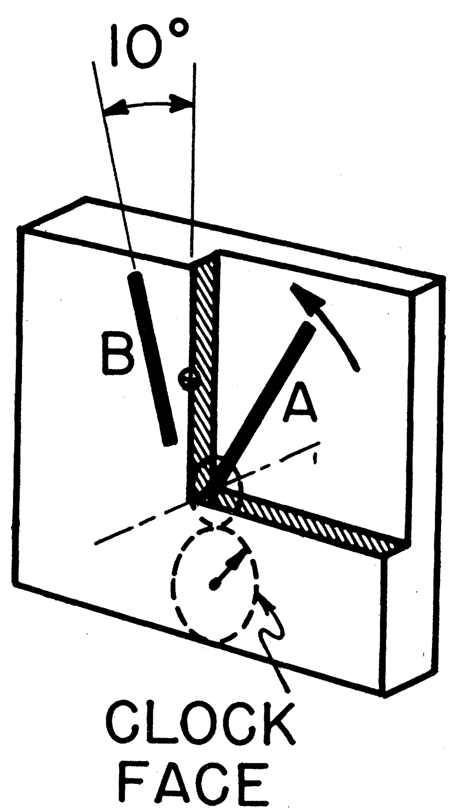

Fig. 1. Apparatus for measuring apparent speed. *A*, Rotating sweep; *B*, fixed marker.

apparent speed. According to this reasoning, even greater reduction should result from exposing observers to a barrage of randomized visual stimulation (extrinsic noise). An electronic device was used to produce a continually changing display of dots on the face of a 14-inch television tube. The resulting pattern may be called visual noise and defined as a changing pattern of dots devoid of redundancies of position or intensity (noisy exposure field). At the opposite extreme from visual noise there is stimulation with minimal temporal variation—a condition that can be produced by exposure to a fixed pattern, called here a hyperstable field because of the absence of even that amount of object-movement found in everyday scenes. To achieve comparability of exposure conditions we used a still photograph of the picture-tube display which showed the set of dots available at one instant (hyperstable exposure field). Such stimulation may suppress intrinsic noise to a degree greater than normal and hence cause an increase in apparent speed.

Finally, the observers were kept alert in total darkness for a comparable period (dark exposure field). Under the latter three test conditions, illumination level and field size (except for the dark exposure field), exposure time, and other controlled variables were equated with those of the first test. The order of presentation was varied for the 12 observers. The resulting changes in apparent speed are shown in Table 1. An analysis of variance showed that the differences among conditions were significant beyond the .001 level.

Thus, a method has been developed for quantifying one consequence of visual deprivation. Tests in which this method was used have yielded results consistent with the following propositions: (i) Simulation and enhancement of an aftereffect of visual deprivation through exposure to a noisy visual field implies that deprivation entails the randomization of sensory-neural activity rather than the diminution or absence of such activity. (ii) Production of an aftereffect opposite to that of deprivation, after exposure to a hyperstable field, implies that typical exposure fields have relevant noise characteristics somewhere between those of the noise used and those of the hyperstable field. (iii) The normal stability of speed perception depends upon continuous exposure to the typical environment.

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Protection by Sulfur Compounds against the Air Pollutants Ozone and Nitrogen Dioxide

Abstract. Two distinct but related pathways of protection against the lethal effects of ozone and nitrogen dioxide are shown by (i) simultaneous inhalation of compounds that furnish –SH or –SS–, or both, and (ii) by injection of thiourea derivatives several days prior to exposure to these oxidant gases. The mechanism of (i) is believed similar to that proposed for the action of radiation-protective compounds; that of (ii) involves the development of a tolerance initiated by the thiourea against the oxidants.

Ozone (O_3) and nitrogen dioxide (NO_2) are potent respiratory irritants which may be injurious when inhaled (*1, 2*), and evidence exists that the concentration of O_3 in urban air is damaging to plants (*3*). Nitrogen dioxide, in addition to acting as a pulmonary irritant, acts as a precursor of O_3 in oxidant smogs by photochemical dissociation (*4*).

In tests designed to determine the acute toxicologic effects of combinations of either known or suspected air pollutants, we found that certain sulfur compounds were effective in counteracting the toxic action of O_3 and NO_2. Partial protection by ascorbic acid against acute lethal effects of O_3 has been previously reported from our laboratories (*5*) as well as by Mittler (*6*), who also found slight protection by sodium thiosulfate. Mittler's conclusion that "compounds which protect against death by radiation . . . are not effective against ozone poisoning" is not supported by our work, however. Tests involved the inhalation exposure of mice to an approximate LC_{50} of either O_3 or NO_2 simultaneously with one or more sulfur compounds. In some instances the sulfur compounds were administered intraperitoneally prior to inhalation of the oxidant. Mortality resulting from a 4-hour exposure, as compared with the mortality from a control exposure to oxidant alone, reflected enhancement or suppression of O_3 or NO_2 toxicity as influenced by the sulfur compound. Methods of generation and analysis of O_3 and vapor concentrations of sulfur compounds have been described elsewhere (*5, 7*). Gaseous sulfur compounds and NO_2 were similarly administered from cylinders in metered amounts and diluted to the desired concentration with purified air. The Saltzman method was used for NO_2 analysis (*8*).

The data in Table 1 represent selected results of a series of tests which show the maximal protection found for each sulfur compound. Comparison of mortalities in sulfur-treated versus control groups indicates the degree of protection afforded. Hydrogen sulfide (H_2S) gave significantly greater protection against oxidant exposure, particularly NO_2, than other sulfur compounds. On a molar basis H_2S protected in a ratio of 1/55 moles of NO_2, whereas benzenethiol, next in order of effectiveness, protected in a ratio of 1/5.4. Higher molar ratios of sulfur compounds were required for protection against O_3, however; for benzenethiol 1.5 mole/mole of O_3, and for H_2S, 2 mole/mole of O_3.

Table 1. Effect of sulfur compounds on the toxicity of nitrogen dioxide and ozone for mice. Mortality at 24 and 72 hours is indicated by deaths/number of mice tested.

Sulfur compound	Av. concn. (ppm)	Av. concn. of oxidant (ppm)		Mortality			
				S treated		Oxidant alone	
		NO_2	O_3	24 hr	72 hr	24 hr	72 hr
1–Hexanethiol	145	78		0/20	1/20	10/20	11/20
1–Hexanethiol	115		4.1	1/20	2/20	10/20	10/20
Methanethiol	65		4.8	2/15	2/15	9/15	9/15
Dimethyl disulfide	45	83		5/20	5/20	10/20	11/20
Dimethyl disulfide	21 mg/kg*	80		4/20	4/20	13/20	13/20
Dimethyl disulfide	21 mg/kg*		4.6	2/20	2/20	10/20	12/20
Hydrogen polysulfide	20 mg/kg*	105		8/25	8/25	16/25	18/25
Di-*tert*-butyl disulfide	24	84		4/15		9/15	
Benzenethiol	14	76		1/20	1/20	10/20	10/20
Benzenethiol	9		6.1	1/20	3/20	11/20	12/20
Hydrogen sulfide†	11		4.9	7/35	7/35	17/35	18/35
Hydrogen sulfide†	1.5	82		1/20	2/20	10/20	10/20
Thiophene	180	85		9/15		8/15	
Dimethyl sulfide	195		4.6	14/30		14/30	

* Administered by intraperitoneal injection. † An aged technical grade.